Lactose Intolerance in Infants and Children

Digestion and Absorption of Lactose

Lactose intolerance is the inability to digest significant amounts of lactose that is present in milk. Lactose is a disaccharide comprised of glucose and galactose and is the main form of carbohydrate in all mammalian milks (Kerner 1995). Its content in human breast milk is independent of the lactose content in the maternal diet. It is present at a constant level throughout a feed and throughout the day (Lawlor-Smith 1998). Lactose is hydrolyzed by the brush-border enzyme, lactase, into the two monosaccharides that are then absorbed into the cytosol via active-sodium transport. In addition, lactose is thought to enhance the absorption of a number of minerals including calcium, magnesium, and zinc (Ziegler & Fomon 1983). Term infants have the full amount of adult lactase activity. The age at which lactase activity in premature infants approximate that of term infants is noted to be about 36 weeks gestation (Antonowics & Lebenthal 1977). Another more recent study found that premature infants of 34 weeks' gestation have 40% of the lactase activity of term infants (Lebenthal et al 1983). Therefore, infant formulas for the non-breastfed premature infants are reduced in lactose and replaced by glucose polymers, because maltase and glucoamylase activity is much higher than is lactase activity in such infants. Lactase activity is developmentally regulated but early enteral feedings have been noted to have a marked effect on the development of lactase activity of premature infants 26 to 30 weeks' gestation (Shulman et al 1998). In addition, these researchers collected data that demonstrated greater lactase activity in premature infants at 10 days of age who received human milk exclusively versus infants who were fed formula. Whether the effects of milk on lactase activity were due to the greater concentration of lactose in human milk compared with that in formula must be determined.

Symptoms

Lactase activity is highest at the tip of the small intestinal villus and is quite low at the base of the villus (Barnard 1997). Because the villus tip is the region most often injured by infection and other pathological processes, lactase activity is commonly decreased. The first enzyme to be affected and the last to recover completely is lactase. Any lactose that is not absorbed will pass undigested into the large bowel. There may be several consequences to the undigested lactose (Lawlor-Smith 1998):

1. Water is drawn via osmosis into the large bowel.
2. Bacteria present in the colon ferment the lactose producing:
    - Short-chain fatty acids including lactic acid which can be absorbed and used for energy
    - Gases such as carbon dioxide, methane, and hydrogen; some of which is excreted through the lungs (giving rise to a positive breath hydrogen test).
3. Stools are therefore:
    - Liquid because of extra water
    - Acid because of unabsorbed fatty acids (pH of stool <6.5 is positive Clinitest – Barnard 1997)

- Positive for reducing substances because of unabsorbed lactose (Clinitest method also).

Symptoms of lactase deficiency are summarized as abdominal distention/bloating, flatulence, abdominal pain from excessive production of hydrogen gas, and osmotic diarrhea from the production of organic acids and low pH. Borborygmi, failure to thrive, and aversion to foods containing the offending carbohydrate may also be observed (Kerner 1995). These symptoms begin to occur about 30 minutes to 2 hours after eating or drinking foods containing lactose (NIH 1994).

Classifications and Dietary Treatments of Lactose Intolerance

Lactose intolerance is divided into the following classifications as described specifically by Kerner1995.

1. **Primary Lactose Intolerance: Irreversible abnormalities**

a. **Congenital lactose intolerance** is extremely uncommon. Dietary treatment of congenital lactase deficiency requires complete elimination of lactose from the diet. Early detection is crucial and suspected in neonates with congenital diarrhea, poor weight gain, and acidic stools with reducing sugars (Barnard 1997). Lactose-free formulas are the formulas of choice, soy-based the most common and least expensive or hydrolysate formulas (Alimentum, Nutramigen, Pregestimil). Cow's milk lactose-free formulas are another option (Enfamil Lactofree and Similac Lactose-free). Because the latter contain trace amounts of galactose, it is advised that the monosaccharide deficiency – galactosemia - be ruled out before it is prescribed to the affected infants. It is of interest to note that lactose-hydrolyzed human milk have been used for infants with congenital lactase deficiency (Simila et al 1982).

b. **Acquired lactose intolerance or "late onset" lactose intolerance** is manifested typically by a *gradual* decline of enzyme levels that begin in childhood. The earliest that late onset lactase deficiency can be present is after the age of 2 years. However, many people may not experience symptoms until they are much older (NIH 1994). Approximately 15% of white adults, 75% to 81% of African-American adults, 75% of Native American adults, and 90% to 100% of Asian adults are lactase deficient (NIH 1994, Simmons 1978). The occurrence of late-onset lactose intolerance is most unusual before the age of 5 in any population and unusual before the age of 10 in whites (Welsh et al 1978). A slight contrast to another study that cites older children and adolescents of most ethic backgrounds except Northern Europeans develop late onset lactase deficiency between 3 and 5 years of age (Angelides 1985).

No treatment exists to improve the body's ability to produce lactase, but symptoms can be controlled by restriction of lactose. It is important to note that the degree of restriction varies from patient to patient which is often learned on a trial and error basis. Consultation with a doctor or nutritionist is advised to educate the parent/guardian on lactose containing foods, tips on reading food-labels for hidden lactose, reduced-lactose milk, and lactase enzymes that are available without

prescription. Lactase enzymes exist in both pill form for individual intake or liquid form for the direct treatment of standard cow's milk. Very young children with the acquired lactose intolerance, incidence rare as it may be, may want to restrict all lactose-containing foods and beverages to avoid osmotic diarrhea.

2. **Secondary Lactose Intolerance: environmentally produced and usually reversible**. By far, the most common cause of lactase deficiency in the first few years of life is secondary lactose intolerance. A variety of common pediatric intestinal disorders may sufficiently injure the small intestinal villus to cause a transient lactase deficiency, namely viral and bacterial gastroenteritis, milk-protein enteropathy, gluten enteropathy, giardiasis, inflammatory bowel disease, and necrotizing enterocolitis (Barnard 1997, Kerner 1995). Rotaviral enteritis is encountered most frequently w/ children less than 3 years of age (Barnard 1997, Guarino 1997).

Refeeding after acute gastroenteritis remains controversial. The relative risk of malnutrition from depleted caloric intake, the temporary lactose intolerance that may cause osmotic diarrhea compounding the risk of dehydration or chronic diarrhea and failure to thrive are serious concerns. On the other hand, continued enteral feedings (oral or tube) prevents malnutrition and therefore may lessen the severity of future episodes of acute diarrhea (Brown & MacLean 1984). In addition, continued enteral feedings stimulate the repair or regeneration of the intestinal mucosa (Sinden & Sutphen 1991). Despite the "malabsorption" seen in acute gastroenteritis, considerable nutrient absorption continues to occur (Brown et al 1988).

The management of the breastfed infant who has acute gastroenteritis should take into account the advantages of breastfeeding in resolving the gastroenteritis and preventing future incidences (Khin-Maung-U et al 1985, Tolboom et al 1986). It can reduce the number and volume of diarrheal stools relative to those seen with cow's milk formula when it is used with an oral rehydration solution. The management of the breastfed infant should include the assurance that the infant is thriving by reviewing the basics to include good positioning and attachment (Lawlor-Smith 1998). Low fat feeds result in rapid gastric emptying leading to large quantities of lactose being presented to the small intestinal brush border. Thus the ability of lactase to digest the lactose may be overwhelmed. The amount of fat being consumed at any feed should therefore be maximized to delay gastric emptying. This can best be achieved by optimizing hindmilk intake. A consultation with a lactation consultant, WIC program, or La Leche League leader may also assist in these matters.

For the bottle-fed infants, the degree of acute gastroenteritis may be a relevant factor to the choice of dietary treatment. For the previously healthy, well-nourished infants with mild acute gastroenteritis, cow's milk formula may be safely used after initial rehydration with a glucose-/rice-electrolyte solution (Sinden & Sutphen 1991, Kerner 1995). Additional studies provide further recommendations on the "how to's" to re-introducing cow's milk formula. A study (Placzek & Walker-Smith 1984) that compared two feeding regimens following acute gastroenteritis concluded that children older than 9 months could be given full-strength cow's milk formula immediately after 24 hours of treatment

with a oral rehydration solution. In children younger than 9 months, however, milk should be reintroduced over a period of 3 to 4 days. In a study by Armitstead et al 1989, infants under 9 months of age with mild acute gastroenteritis were randomized to gradual reintroduction of their normal milk, immediate return to full-strength formula, or a rapid regrade to a hypoallergenic whey hydrolysate formula. The process of slow regrade was as follows: oral carbohydrate-electrolyte solution for 24 hours → quarter-strength feeds → half-strength feeds → three-quarters strength feeds → full-strength feeds within next 12-24 hours. There was no significant difference in relapse rates between these groups. In addition, no child developed lactose intolerance. All relapses were due to return of diarrhea without evidence of carbohydrate intolerance. The Armitstead study suggests that there is no need to regrade and that significant lactose intolerance is now uncommon. In developed communities, the incidence of lactose intolerance after acute gastroenteritis is significantly diminishing (Kerner 1995).

It is Penny and Brown 1992 who identified three simple clinical characteristics of Peruvian children who would deteriorate if the children continued to consume a diet containing 6 g lactose/kg body weight/day and 110 kcal/kg/day. They were young age (< 12 months), malnourished status (weight-for age <2 SD below NHCS norms), and presence of fever.

Secondary lactose intolerance is often a component of chronic gastrointestinal disease in children. Disorders such as gluten-sensitive enteropathy, Crohn's disease, ileitis, and short-gut syndrome, or other conditions leading to villous atrophy, may result in decreased lactase activity (Pironi et al 1988). The degree of restriction varies from individual to individual. Lactose restriction in people with chronic gastrointestinal disease may also be temporary until lactase activity increases after the resolution of the primary mucosal lesion (Angelides 1985).

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